PB 188 086

AIR POLLUTION ASPECTS OF CADMIUM AND ITS COMPOUNDS

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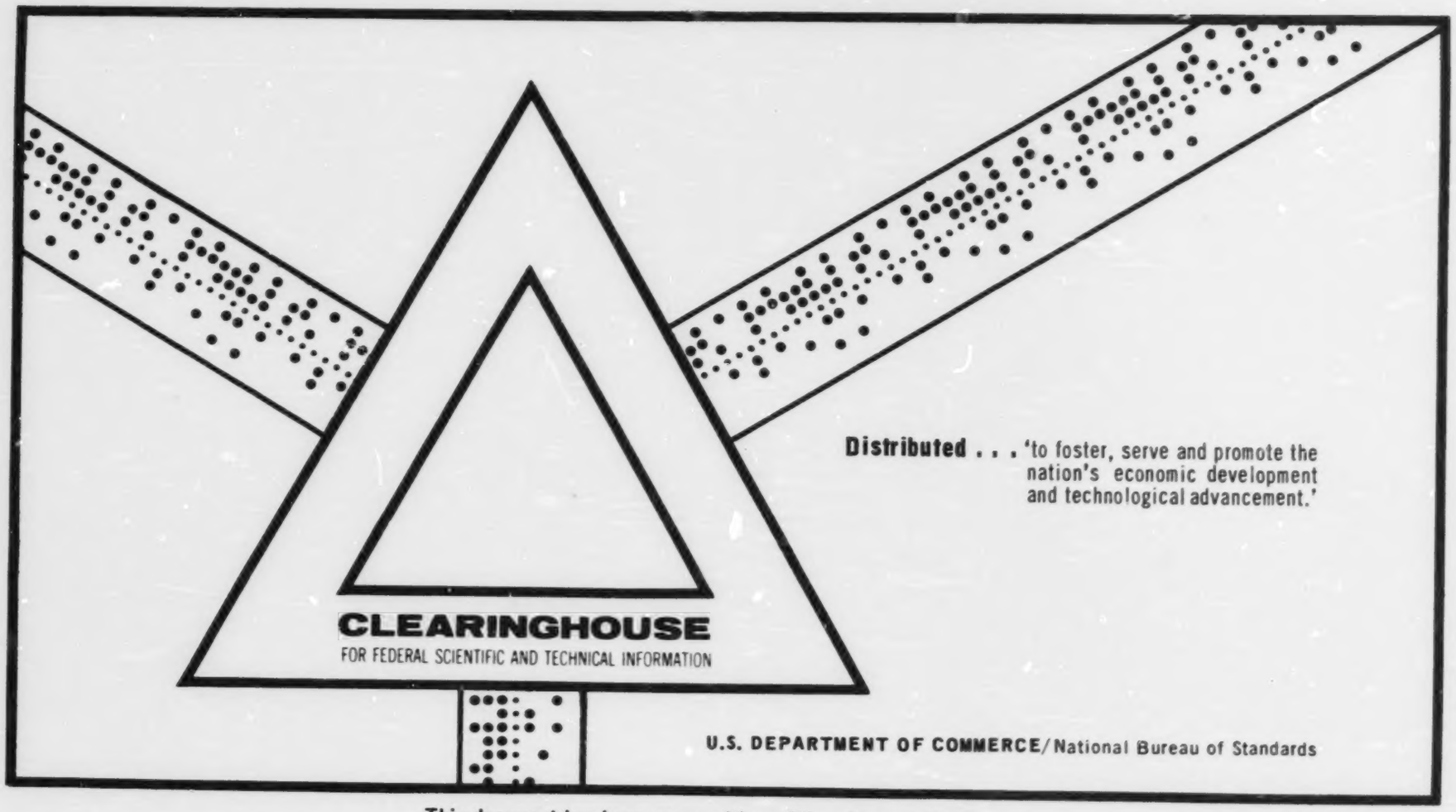
September 1969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AIR POLLUTION ASPECTS

OF

CADMIUM AND ITS COMPOUNDS

Prepared for the
National Air Pollution Control Administration
Consumer Protection & Environmental Health Service
Department of Health, Education, and Welfare
(Contract No. PH-22-68-25)

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September 1969

FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

Aeroallergens (pollens)
Aldehydes (includes acrolein and formaldehyde)
Ammonia
Arsenic and Its Compounds
Asbestos
Barium and Its Compounds
Beryllium and Its Compounds
Biological Aerosols (microorganisms)
Boron and Its Compounds
Cadmium and Its Compounds
Chlorine Gas
Chromium and Its Compounds (includes chromic acid)
Ethylene
Hydrochloric Acid
Hydrogen Sulfide
Iron and Its Compounds
Manganese and Its Compounds
Mercury and Its Compounds
Nickel and Its Compounds
Odorous Compounds
Organic Carcinogens
Pesticides
Phosphorus and Its Compounds
Radioactive Substances
Selenium and Its Compounds
Vanadium and Its Compounds
Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are

necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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Appreciation is expressed to the many individuals both outside and within NAPCA who provided information and reviewed draft copies of these reports. Appreciation is also expressed to the NAPCA Office of Technical Information and Publications for their support in providing a significant portion of the technical literature.

ABSTRACT

Cadmium and cadmium compounds are toxic to humans and animals; however, the exact nature of the biological action of cadmium is not fully understood. It is known to be toxic to practically all systems and functions of human and animal organisms, and it is absorbed without regard to the level of existing concentrations in the body, indicating the lack of a homeostatic mechanism for the control of cadmium levels.

When inhaled, cadmium or cadmium compounds can produce pulmonary emphysema and bronchitis, kidney damage resulting in proteinuria, and gastric and intestinal disorders. Atmospheric concentrations of cadmium have been related statistically to diseases of the heart. Similar effects have been observed in experimental animals. Effects on the liver and brain have also been observed. Animal experiments also indicate that cadmium may be carcinogenic.

No evidence has been found of the deleterious effects of cadmium air pollution on plants or materials.

The major emission sources of cadmium are the metal industries engaged in extraction, refining, machining, electroplating, and welding of cadmium materials. Since cadmium is produced as a by-product rather than as a primary product, the refining of associated metals—such as zinc, lead, and copper—provides the emission sources important to air pollution. Cadmium is also used in the manufacture of pesticides and

fertilizers, and both the production and consumption of these products are potential air pollution sources.

The average ambient air concentration of cadmium was 0.002 $\mu g/m^3$ in 1964, and the maximum concentration was 0.350 $\mu g/m^3$.

General air pollution control procedures used in the metal industries are applicable to the abatement of cadmium contamination; however, little information has been found on specific applications of these procedures.

No information has been found on the economic costs of cadmium air pollution or on the costs of its abatement. Methods are available for the analysis of cadmium in the ambient air.

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1. INTRODUCTION

Cadmium is a relatively rare element discovered in 1817 by Strohmeyer, who gave it the name "cadmium" to indicate its close association with zinc ores.

The industrial production of cadmium started in 1829 in Upper Silesia and in 1907 in the United States. Cadmium production in the United States has been accelerating since the beginning of the 20th century. The cadmium industry's steady growth is a result of an increasing number of industrial uses—such as electroplating—and products—such as storage batteries, semiconductors, atomic fission controls, fertilizers, and pigments.

Industrial exposure to cadmium has been observed to cause both chronic and acute poisoning. Inhalation of cadmium fumes or vapors causes damage mainly to kidneys, where the cadmium tends to concentrate. Moreover, most other body organs and systems can be affected adversely by cadmium inhalation.

The most likely source of cadmium pollution is the production and the handling of its compounds. The inhalation of fumes and vapors arising from retorts or condensers while cadmium and cadmium oxide are being manufactured, and the inhalation of dust in bagging have caused industrial cadmium poisoning.

Recent studies indicating that cadmium may cause heart disease and cancer have caused concern about the health significance of low concentrations of cadmium in ambient air.

2. EFFECTS

2.1 Effects on Humans

Although human exposure to concentrations of cadmium in the atmosphere has occurred for over 140 years, no systematic study of the adverse effects of such exposure has yet been made. Nevertheless, studies conducted during the last 10-year period have indicated that there is no system or function of the human organism that has not been subjected to and damaged (experimentally or otherwise) by an effective concentration of environmental cadmium.

Table 10 in Appendix B summarizes the specific effects noted from intake of cadmium or cadmium compounds by humans. Table 11 in Appendix B indicates the cadmium content of various human tissues, as reported by Schroeder, et al.[91] The properties and toxicity of certain cadmium compounds are presented in Table 12 (Appendix B). Appendix A provides additional information on the physical and chemical properties of cadmium and some of its compounds.

2.1.1 Respiratory System

Pulmonary emphysema—the most typical symptom of chronic cadmium poisoning due to inhalation of cadmium fumes, cadmium oxide dust, or cadmium salts—is characterized by shortness of breath and may be followed by bronchitis. In pulmonary emphysema the alveoli of the lungs are dilated and their walls overdistended.[12]

The effects of inhaled cadmium on the respiratory system are characterized by three overlapping phases: edematous, proliferative, and fibrogenic.[27] The first phase, representing the initial inflammation of some 50 square meters of irritated pulmonary membrane, is similar in nature to that produced by other deep-lung irritants. Interstitial edema develops as a result of extravasation of fluid from inflamed pulmonary capillaries and often results in a separation of pulmonary epithelium sheets from their underlying stromata. Progressive distention of interstitial tissues is followed by accumulation of fluid in the alveoli, and often, precipitation of fibrin. The second phase is characterized by metaplasia of the alveolar epithelium and proliferation of histiocytes and polypoidal lesions, resulting in destruction of alveoli and proliferative interstitial pneumonitis. The third, or fibrogenic, phase has been observed in experimental animals surviving acute cadmium exposures and also among exposed humans who survived. The associated pulmonary fibrosis is localized around the bronchi and the vascular tubes.

Clinical examinations of five workers poisoned by cadmium fumes while cutting cadmium-plated bolts with an oxyacetylene torch showed severe pulmonary edema and alveolar metaplasia of the lung.[10]

2.1.2 Cardiovascular System

Perry and Schroeder,[71] and later Schroeder alone,[87,88] implicated cadmium as a cause of hypertension. Their studies

showed a correlation between the cadmium content of the kidneys and hypertension in both humans and rats. Stimulated by these results, Carroll[17] compared the death rates in 28 U.S. cities and the cadmium concentration in the urban air. He found a significant correlation (r=0.76) between the cadmium concentration and diseases of the heart (with the exception of rheumatic heart disease). His study included hypertension, arteriosclerosis, and other nonspecific heart diseases, such as congestive heart failure and myocardial degeneration.

In another study that related diseases of the heart to concentrations of cadmium, zinc, lead, vanadium, tin, chromium, and manganese in the urban air, cadmium concentration was found to have the highest correlation to heart diseases, followed by vanadium.[49]

In a study of 13 Japanese workers engaged in smelting silver-cadmium alloys, exposure for even less than a year to the resulting cadmium fumes (75 to 240 $\mu g/m^3$, averaging 130 $\mu g/m^3$) produced anemia.[100]

2.1.3 Other Physiological Effects

Kidney damage is the most common and the best documented of the effects of cadmium on the excretory system of human and animal bodies.

Prolonged occupational exposure to cadmium dusts and fumes produces proteinuria, i.e., excretion of urinary proteins with molecular weights of 20,000 to 30,000. Documentation

relating cadmium exposure to proteinuria is relatively extensive and detailed.

Friberg[35] observed proteinuria in Swedish workers who had been engaged in the alkaline battery industry. These workers had experienced comparatively short exposures (5 to 7 years) to low concentrations (200 to 15,000 $\mu g/m^3$) of cadmium dust. In one case, the proteinuria was detected when the patient complained of lassitude 3 years after he had ceased handling the cadmium.

A clinical study of 70 workers exposed to cadmium oxide dust was reported by Potts.[77] These workers were exposed to cadmium concentrations ranging from 100 $\mu g/m^3$ to 236,000 $\mu g/m^3$ over a 10-year period. Proteinuria and anosmia were found in 31 (44 percent) and 45 (64 percent), respectively. Potts reported two cases of glycosuria, six of bronchitis, six of peptic ulcers, and eight deaths. Five of the eight deaths were caused by cancer (three from cancer of the prostate). He makes no claim that these findings are causally associated with cadmium exposure.

Japanese workers who had been exposed to dusts of stabilizers (including cadmium stearate) in a vinyl chloride film factory were found to have a high incidence of proteinuria.[97]

Another study of proteinuria due to cadmium oxide fumes involved 79 persons, 55 of whom were workers exposed to the fumes. Piscator[73] found that normal protein excretion in the

urine averaged 50,000 µg per day, while in the individuals exposed to cadmium oxide fumes, protein excretion ranged from 70,000 to 2,600,000 µg per day. Furthermore, this excretion rate was found to be a function of the length of exposure. The electrophoretic findings of this study were typical of tubular proteinuria.

Quantitative determinations of proteinuria have shown that the appearance of proteinuria is not a specific indication of cadmium exposure, as other diseases give rise to similar reactions.[74]

Piscator[75] also compared normal urinary proteins with excreted proteins from patients with occupational proteinuria caused by chronic cadmium poisoning: most of the excreted proteins were identified as normal urinary proteins. The study concluded that the presence of large amounts of low molecular weight (20,000 to 30,000) serum proteins in the urine of patients with chronic cadmium poisoning is probably due to tubular damage and the resulting reduction in efficiency of protein absorption in the glomerular filtrate.

Another study reported that the autopsy of a worker poisoned by cadmium fumes while cutting cadmium-plated bolts with an oxyacetylene torch showed renal damage.[10]

Nephrolithiasis was observed in about 25 percent of the workers in the Swedish alkaline battery industry.[33,37]

Kidney damage is now considered to be a classical syndrome of chronic cadmium poisoning, and it has been shown

to result from exposure to the soluble salts of cadmium sulfide and cadmium stannate.[55]

The variation, with respect to age, of cadmium mean concentrations and of the cadmium-to-zinc ratio found in kidneys by Schroeder et al.[91] is shown in Figure 1. Maximum concentrations of cadmium and cadmium to zinc were found in the 40-to-50-year age group. Concentrations declined thereafter.

Other observed and documented physiological effects of cadmium include gastric and intestinal disorders and damage, loss of weight, and hepatic damage.

Sakabe and Ushio[85] found gastric disturbances caused by inhalation of cadmium stearate dust. They suspected that the inhaled dust (1 to 10 μ*) was deposited in the upper respiratory tract where the particles would be removed by ciliar motion and expectoration, then swallowed and hydrolyzed by the acidic gastric juice.

Potts[77] reported that six men exposed to cadmium oxide dust had peptic ulcers, of whom four have had partial gastrectomies.

2.1.4 Carcinogenesis

There is incomplete evidence that cadmium is carcinogenic to humans, however, its carcinogenicity in animals has been demonstrated.

In 1965, Potts[77] reported that from a total of 70 cadmium

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*μ=micron(s).

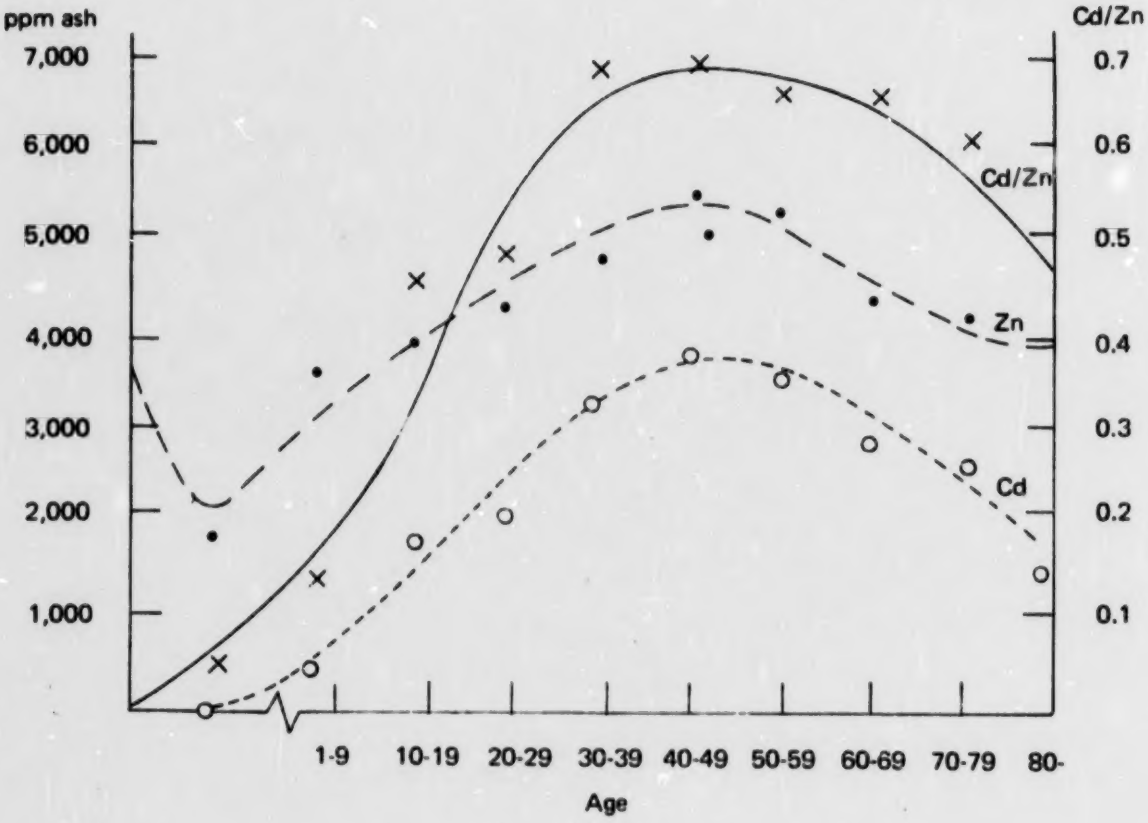

FIGURE 1

Mean Concentrations of Cadmium and Cadmium/Zinc Values in Kidneys of 221 Subjects from the United States, According to Age[91]

workers five deaths were due to cancer. In the discussion that followed the presentation of this paper, Kazantzis suggested that these results indicate a possible role of cadmium as a carcinogen. He further pointed out that some cadmium compounds have been shown to be carcinogenic to animals (see Section 2.2.2.3).

In another study[14] ten different tissues obtained from the Autopsy Service of the Los Angeles County Hospital were analyzed for 13 trace metals. Cadmium concentrations in cancerous tissues obtained from lung and liver were found to be nearly twice as high as those obtained from control tissues (4,000 and 5,100 vs. 2,400 and 3,200 µg/100 g dry tissue).

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

A few studies have been conducted on the effects of cadmium that has been injected into farm animals or included in their diets. Although these studies are not directly related to effects of cadmium in the environmental air, they serve to assess the relative toxicity of cadmium to various species of animals.

In a study by Miller *et al.*[65] three cows were each given capsules of 3 grams of cadmium daily for 2 weeks. Milk production declined rapidly for several days after cadmium administration was begun, and then increased to an intermediate level. When cadmium administration ceased, milk production increased by 50

percent and weight loss occurred. Fecal excretion of cadmium averaged 82 percent of the intake. The cadmium content of milk was less than 0.1 ppm, which was the low detectability limit. Less than 0.022 percent of the cadmium administered appeared in the milk.

Lofts et al.[60] reported that an intramuscular injection (0.2 ml of a 0.04 molar solution) of cadmium chloride given to wood pigeons and feral pigeons did not result in necrosis of the seminiferous tubes. However, they found that an intratesticular injection (0.1 ml of a 0.04 molar solution) of cadmium chloride into pigeons of both species caused localized necrotic damage and a general disruption of germ-cell coordination in the seminiferous epithelium.

A study[5] was made of cadmium toxicity in swine by feeding a ration containing a wormer (0.044 percent cadmium anthranilate) to shoats in dry lot. Some deaths occurred: autopsies revealed anemia, enlarged spleen, numerous linear ulcerations of stomach, enlarged mulberry-shaped heart, marked fatty degeneration of liver and kidneys, and icterus. Withdrawal of the medicated feed eliminated the symptoms.

2.2.2 Experimental Animals

A most important category of health effects is one characterized by complete absence of definite symptoms. Effects on longevity and survivability of living organisms belong to this category and can be causally associated with a

pollutant only by extensive experiments employing refined and well-designed statistical methods of analysis.

Schroeder et al.[90] showed that the life span of rats and mice fed cadmium was shorter than that of control groups. In this study 127 rats fed cadmium were compared with 104 control rats. The findings of the study are summarized in Table 1.

TABLE 1

LIFE SPAN OF RATS[90]

Percent Dead	Age (days)	
	Control	Cadmium-fed
50 percent	945-978	805-822
75 percent	1,016-1,036	902-996
100 percent	1,185-1,400	1,213-1,278
Number of dead at 3 months of age	0	20

Data on some effects of specific doses of cadmium compounds on animals are listed in Table 13 in Appendix B.

2.2.2.1 Respiratory System

Schroeder et al.[90] found no significant difference in the number of deaths caused by pneumonia in rats fed 5 ppm cadmium acetate solution and a control group. These investigators observed at autopsy that 26 rats (or 31 percent) from a total of 84 died of pneumonia while being fed cadmium. In comparison, 25 (or 36 percent) of 70 rats in a control group died of pneumonia.

In another study,[101] rats intermittently exposed to cadmium chloride aerosol developed larger lungs, which contained an air volume three times greater (1.5 ml) than the normal; a lung-to-body weight ratio markedly greater (86.5 x 10^{-4}) than the normal (4.2 x 10^{-4}); and a decreased amount of air per gram of body tissue. The last symptom indicates loss of distensibility, or increased rigidity. Both macroscopic and microscopic examinations showed pulmonary inflammation, fibrosis, emphysema, loss of tissue elasticity, decrease of functioning units (decrease in the number of alveoli), and filling of alveoli with proliferated cells and debris.

2.2.2.2 Other Physiological Effects

A dose of 650 µg/kg of cadmium injected as cadmium sulfate in rabbits was found by Dalhamn and Friberg[23] to result in a 40 percent to 50 percent reduction in hemoglobin within 10 weeks, as well as a 30 percent to 50 percent reduction in erythrocytes. As a consequence, the rabbits developed pronounced anemia.

In a subsequent study, Carlson and Friberg[16] showed that rabbits injected daily for 1 to 6 weeks with solutions of radioactive cadmium-115 (650 to 1,000 ug/kg body weight) developed anemia. The erythrocytes contained more than 90 percent cadmium in whole blood, and the cadmium in the blood, after repeated exposures, was at least partly bound to the hemoglobin molecule. The study concluded that it is possible

that cadmium is transported to the bone marrow, where it inhibits the synthesis of hemoglobin and thus produces anemia in animals and men.

In another study, Dalhamn and Friberg[22] injected cats and rabbits with very small doses of cadmium (300 to 500 µg of cadmium per kilogram of body weight) in the form of cadmium sulfate solution. A reduction in blood pressure of 15 to 45 mm Hg was observed almost immediately. The blood pressure returned to normal in 1 to 3 minutes.

An investigation by Kennedy[57] of the effect of injected cadmium chloride (10, 15, and 20 µ mole/kg body weight) on the serum calcium levels of rabbits showed hypocalcemia proportional to the cadmium dose. The experiments showed that the serum calcium level dropped from 147 µg/ml to 100 to 120 µg/ml due to increased renal excretion.

Dalhamn and Friberg[23] found that a dose of cadmium (650 µg/kg) injected subcutaneously in rabbits resulted in proteinuria in 16 of the 18 exposed animals in about 1 to 2 months after injections were begun.

Later they reported in a supplementary study[24] (on 18 rabbits injected with 650 µg cadmium per kilogram of body weight in the form of a water solution of cadmium sulfate) that microscopic examination of the sacrificed animals showed pronounced damage to the renal tabuli.

Prolonged administration of small doses of soluble cadmium salts to rats was shown to produce renal lesions,

tabular atrophy, and interstitial fibrosis. The lesions were found to be very similar to those found in the case of renal failure in man.[90]

In a study by Schroeder et al.[90] from a total of 46 rats fed cadmium, 35 animals suffered arteriosclerosis, 13 showed glomerular changes, and three had pyelonephritis.

Schroeder et al.[90] also reported that upon microscopic examination, 16 of 20 rats fed cadmium acetate suffered hepatic sclerosis.

An investigation of rats fed cadmium stearate for 90 days (0.03 percent cadmium diet) revealed histological changes in the stomach and the intestines. It was also shown that all test animals exhibited growth suppression.[104]

A dose of 650 ug metallic cadmium injected in rabbits was found to result in loss of weight, splenic fibrosis, and nephrosis in nearly all the exposed animals.[23]

An increasing number of investigations have been focusing on the effects of exposure to cadmium concentrations on the reproductive system in general, and the function of the orchis in particular.

In one study, rats fed cadmium stearate (0.03 percent cadmium diet) for 90 days showed histological damage in the orchis.[104] Other studies have focused on the destructive effect of cadmium ions on testicular tissue,[68] the sterilization of the male by cadmium salts,[84] changes in the blood vessels of the rat testis and epididymis produced by cadmium

chloride,[20,53] the effect of chryptorchidism and cadmium on the rat testis,[25] the path of cadmium[91] in the spermatogenic pathway,[42] and the functioning of the testis and intratesticular ovarian grafts in male rats following injection with cadmium.[62] Most of the above-mentioned studies were conducted during the 1960's. Also during this period, two British studies showed an association between cadmium and teratogenesis in animals.[42,84]

Holmberg and Ferm[51] have reported that cadmium sulfate is teratogenic to the hamster embryo. Following injection of 2 µg/g of cadmium sulfate into the lingual vein of pregnant hamsters, 59 of 115 embryos were malformed. Selenium was found to significantly decrease the teratogenic action.

Comparatively very few studies have been made of the effects on the nervous system of exposure to cadmium in the atmosphere.

In a study by Schroeder et al.,[90] of 84 rats fed cadmium acetate (5 ppm), the deaths of 10 amimals were caused by disorders of the nervous system. The major symptoms were vestibular disturbances, paralysis, convulsions, and "rolling fits." In the control group, only one of 70 animals died of neurological disease.

A Russian study of chronaxic and conditioned reflexes of animals exposed to inhalation of cadmium oxide aerosol showed that a single inhalation of 13 to 18 µg/liter as well as repeated exposure to concentrations ranging from 1.8 to 2 µg/liter

produced disturbances of conditioned reflex activity. In both acute and prolonged exposures, similar results appeared after prolonged latent periods and were accompanied by lower reflex responses and, in some cases, hypnotic phases.[101]

2.2.2.3 Carcinogenesis

Rats injected with cadmium chloride solution developed sarcomas at the injection site (mesodermal tissues), while comparable dose levels of zinc failed to produce tumors in any of the tissue sites tested.[41]

Sarcomas were also induced in rats by injections of suspensions of cadmium metal powder,[47,48] cadmium oxide,[56] cadmium sulfide (insoluble),[54,56] and even cadmium sulfate (soluble).[44] Rat ferritin, an iron preparation known to be contaminated with cadmium, produced tumors when injected into rats.[43]

2.3 Effects on Plants

Cadmium reaches the soil as an impurity in phosphate fertilizers.[9] When vegetables normally deprived of cadmium were grown in soil heavily fertilized with 20 percent superphosphate, they absorbed the cadmium, and the roots became progressively damaged.[89] While contamination of soil by atmospheric zinc has been studied, no similar study has been made for cadmium.

2.4 Effects on Materials

No evidence was found in the literature regarding effects

on materials resulting from cadmium air pollution.

2.5 Environmental Air Standards

The American Conference of Governmental Industrial Hygienists[99] has recommended the following cadmium threshold limit values for industrial workers:

Cadmium oxide fumes	100 $\mu g/m^3$
Cadmium metal dusts and soluble salts	200 $\mu g/m^3$

The American Industrial Hygiene Association[15] recommends a maximum atmospheric concentration of 100 $\mu g/m^3$, for cadmium oxide fume. In addition, they suggest that doses of 2,900,000 $\mu g\text{-}min/m^3$ of cadmium oxide can be lethal; 1,100 $\mu g/m^3$ for 8 hours can produce nonfatal intoxication and 500 to 2,500 $\mu g/m^3$ over a 3-day period can produce cadmium pneumonitis.

The U.S.S.R. has adopted the threshold limit value of 100 $\mu g/m^3$ per 8 hours for cadmium oxide for industrial workers.

British Commonwealth countries have accepted the following values for emission standards of cadmium in effluent air or gases:

Great Britain	39,000 $\mu g/m^3$
New South Wales	23,000 $\mu g/m^3$
Queensland	23,000 $\mu g/m^3$

Great Britain also limits the emissions of cadmium to 30 pounds per week (168 hours).[63,95]

3. SOURCES

3.1 Natural Occurrence

Pure cadmium metal does not occur in nature. It is a relatively rare metal that occurs mainly in the form of minerals in zinc, lead-zinc, copper-zinc, and lead-copper-zinc ores. In fact, cadmium is found in nearly all ores containing zinc, and its concentration is almost always in direct relation to that of zinc. The cadmium-to-zinc weight ratio varies from 0.0002 to 0.002.[102] The following concentrations are cited in the literature:[19,92]

Material	Cadmium Concentration
Earth's crust	0.000055%
Geological reserves (percent of zinc reserves)	0.5%
Ores	
Zinc blende	0.1-5.0%
Pribramite	5.0%
Polymetallic zinc ores	0.01-0.5%
Zinc concentrates from sulfide ore	0.3-1.4%
Minerals (primary)	
Greenockite (CdS)	≤77.6%
Octavite ($CdCO_3$) rare	≤61.5%
Sphalerite (CdS coating, crystal)	
Xanthochroite (CdS coating, amorphous)	≤1.47%

The concentration of cadmium in soils has been estimated[91] as follows:

Soil, field, unfertilized	0.55-2.45 ppm (wet weight)
Soil, tilled, unfertilized*	~0.8
Soil, fertilized	3.38

*Virgin Islands, Agricultural Station.

Cadmium concentrations have been determined for the following:[91,61]

Sea water (ppb)	0.075-0.32
Soft waters (µg/liter)	
Natural water	600-14,600[a]
Municipal water	0.0-77,000[b]
Water in private dwellings	1,100-140,000[c]

3.2 Production Sources

As there are no cadmium ores as such, cadmium is produced as a by-product in the refining of other metals, primarily zinc. Cadmium occurs in most of the polymetallic ores, and in the selective flotation of the ores, cadmium accompanies the primary metals and passes into the corresponding concentrates. Therefore cadmium dust, fumes, and mists are commonly present in the refinery processes involving zinc, copper, and lead, as well as in the specific processes for extracting cadmium. These production sources, therefore, are sources of emission of cadmium into the atmosphere. The quantities emitted are a function of the quantities of zinc, lead, and copper produced.[92]

Little information has been found on the specific emissions of cadmium in the metal refinery process. However, it is possible to estimate the amount of cadmium discharged into the atmosphere from data on production and emission of other

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[a]Connecticut River in Vermont.
[b]Stagnant, Brattleboro, Vt.
[c]From tap, plastic pipe; originating in lake in New Hampshire.

metals. Table 2 contains information on the relative amount of cadmium and cadmium compounds contained in dusts from an electrostatic precipitator controlling the roasting process for zinc concentrates.

TABLE 2

CADMIUM CONCENTRATIONS IN ZINC REFINERY DUSTS[19]

Compound	Concentration
BY CHEMICAL ANALYSIS	
Cadmium	0.42%
BY RATIO ANALYSIS	
Cadmium sulfate	0.54%
Cadmiun oxide	0.04%
Cadmium oxide-iron oxide	0.16%
Cadmium sulfide	0.03%

The cadmium content of gases produced in the electrolytic refining of copper are presented in Table 3.

TABLE 3

CADMIUM CONTENT IN EXHAUST GAS OF COPPER SMELTING WORKS[19]

Activity	Cadmium Content of Gas (g/ton)*
Reverberatory smelting	1,500
Blast furnace smelting	2,000
Bessemer process	3,200
Total	6,700

*Grams of cadmium in exhaust gas per metric ton of copper produced.

In 1948 in Pennsylvania, the Donora episode initiated the first detailed measurement of cadmium emission rates in the ambient air from one of the major sources of cadmium, a zinc metallurgical plant. The plant studied discharged a total of 320 pounds of cadmium into the atmosphere per day from the sinter plant and the spelter furnaces.

A study[83] of the Hudson Bay Mining and Smelting Company, Ltd., revealed that prior to an improvement in their methods of recovering cadmium, the amount of cadmium in the dust burden of the smelter stack flow ranged from 0.3 to 0.4 tons/day.

In the United States, the major cadmium plants use as raw material copper-cadmium residues from electrolytic zinc production, lead dusts from lead smelting furnaces, zinc sulfide concentrates, and dust from the elctrostatic precipitators of the multiple-hearth roaster furnaces.

The rapid growth of domestic cadmium production since its beginning (1907) is shown in Figure 2. Data on the production of cadmium, cadmium oxide, and cadmium sulfide for the period 1946 to 1966 are given in Table 4. Table 5 lists the major producers of primary cadmium (as a by-product in the production of zinc).

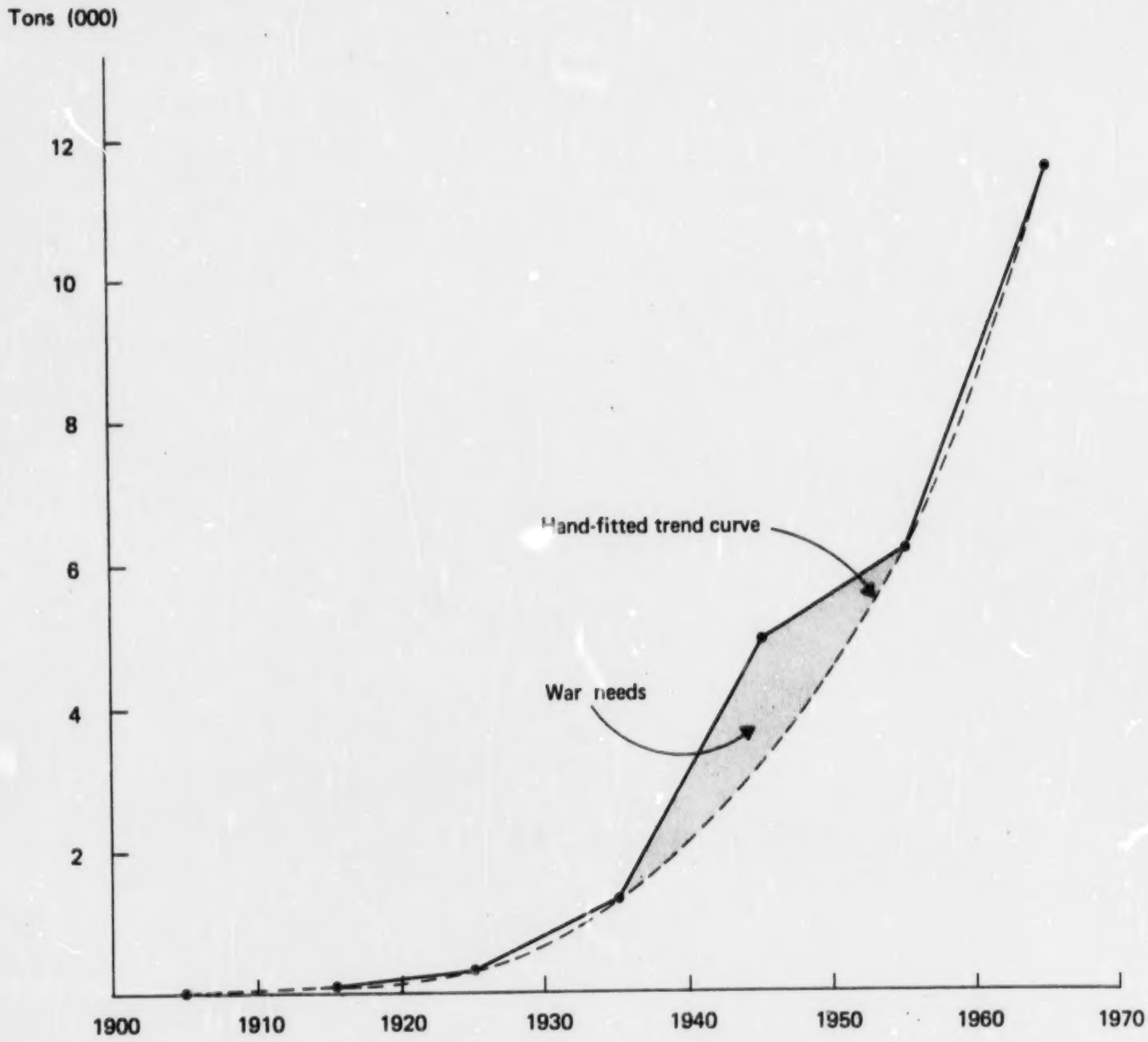

FIGURE 2

U.S. Production of Cadmium
(10-year averages)[66]

TABLE 4

PRODUCTION AND CONSUMPTION OF CADMIUM AND CADMIUM COMPOUNDS[66]
(Thousands of Pounds)

	DOMESTIC PRODUCTION AND CONSUMPTION							
		PRODUCTION						
Year	World Production of Metal	Metal	Oxide	Sulfide	Shipments By Producers	Imports For Consumption	Exports	Apparent Consumption
1966	26,600	10,460	W*	2,267	11,792	3,358	379	14,780
1965	26,100	9,671	W*	1,575	8,128	2,121	73	10,431
1964	28,250	10,458	W*	1,531	9,689	1,104	1,439	9,364
1963	26,000	9,990	W*	1,542	10,124	991	1,313	11,482
1962	25,800	11,137	1,481	1,329	12,057	1,117	717	12,146
1961	25,700	10,466	1,075	1,115	10,222	1,079	702	10,184
1960	21,700	10,180	W*	1,034	12,151	942	2,448	10,166
1959	19,800	8,602	W*	1,243	11,273	1,638	900	11,471
1958	19,800	9,673	W*	983	7,921	1,002	580	8,177
1957	20,800	10,549	W*	1,041	10,091	1,586	693	10,999
1956	20,000	10,614	W*	1,258	10,936	3,116	1,234	12,711
1955	17,920	9,754	W*	1,095	11,453	927	1,394	10,689
1954	15,900	9,552	838	1,045	8,207	402	999	7,424
1953	15,710	9,767	956	1,229	8,282	1,555	66	9,627
1952	13,702	8,567	531	898	8,048	1,479	301	9,008
1951	13,514	8,311	529	955	8,052	90	606	7,171
1950	13,239	9,190	505	1,571	9,620	630	353	9,546
1949	11,506	8,227	498	999	8,455	157	566	7,486
1948	10,728	7,776	292	1,097	7,953	10	956	7,818
1947	10,876	8,508	393	1,308	8,489	20	303	7,727
1946	8,924	6,471	318	1,226	6,812	17	140	6,984

*W = Withdrawn (company confidential).

TABLE 5

MAJOR U.S. PRODUCERS OF PRIMARY CADMIUM
AS A METALLURGICAL BY-PRODUCT OF ZINC[13]

Company	Location
Amax	Blackwell, Okla. (zinc smelter)
Asarco	Denver, Colo. (Globe cadmium plant)
Asarco	Corpus Christi, Tex. (zinc smelter)
American Zinc	East St. Louis, Ill. (zinc smelter)
Anaconda	Great Falls, Mont. (zinc smelter)
Bunker Hill	Kellog, Idaho (zinc plant)
Eagle-Picher	Henryetta, Okla. (zinc smelter)
National Zinc	Bertsville, Okla. (zinc smelter)
New Jersey Zinc	Depue, Ill. (zinc smelter)
New Jersey Zinc	Palmerton, Pa. (zinc smelter)
St. Joseph Lead	Herculaneum, Mo. (lead smelter)

Asarco and Anaconda produce approximately 60 percent of the total output of cadmium in the United States. Amax, American Zinc, and New Jersey Zinc account for an additional 20 percent, while the remaining companies produce the balance.

The domestic production of cadmium has been decreasing in proportion to the world's total production. In 1946, the United States produced 73 percent of the world's production of cadmium. This figure had decreased to 53 percent by 1956, and to a low of 39 percent in 1966.[66] This decrease in the relative domestic supply relates directly to the increased imports of cadmium source materials from Mexico, Peru, and Canada. It also has influenced an increase in efficiency of cadmium recovery from the ore-extraction industries, as revealed in Table 6.

TABLE 6

PERCENTAGE OF CADMIUM RECOVERED PER TON RECOVERABLE ZINC[19]

Year	Percent	Year	Percent
1941	5.2	1948	7.2
1942	5.4	1949	8.2
1943	5.8	1950	9.0
1944	6.7	1951	7.3
1945	6.8	1952	6.5
1946	6.2	1953	8.2
1947	7.2	1954	9.6

3.3 Product Sources

The three major uses of cadmium are in the electroplating process, in alloys and solders, and in pigments and chemicals. The distribution of cadmium uses is tabulated in Table 7 for the years 1940 and 1955-56.

TABLE 7

MAJOR USES OF CADMIUM[19]

Use	Percent of Consumption 1940	1955-56
Electroplating	59.8	58.1
Alloys and solder	23.9	10.9
Pigments and chemicals	10.0	27.3
Miscellaneous	6.3	3.7

Electroplating has consumed the largest portion of the cadmium metal, and almost all of the oxide, sulfate, and chloride produced.

The percentage of cadmium in certain cadmium alloys and solders is listed in Table 8.

TABLE 8

CADMIUM ALLOYS AND SOLDERS AND THEIR CADMIUM CONTENT[96]

Material	Percent of Cadmium
Alloys	
Cadmium-nickel	93.65
Cadmium-silver-copper	97.65
Solders	
Silver-cadmium	95.00
	16.00
	5.00
Zinc-tin-cadmium	22.50
Lead-tin-cadmium	26.00
	22.00
Other-cadmium	40.00
	10-26.00
	8.00

The cadmium content of common paints is indicated in Table 9.

TABLE 9

CADMIUM CONTENT OF PAINTS[78]

Material	Percent of Cadmium
Cadmium red	69.92
Lithopone 30%	0.05
Lithopone 60%	0.05
Stachtolith	0.05
Cadmium red + lithopone 60%	56.09
Cadmium red + lithopone 30%	7.01
Stachtolith + lithopone 60%	0.06

Cadmium is a constituent of fertilizers: common superphosphate fertilizers contain 50 to 170 ppm of cadmium. Cadmium is also used in pesticides. Table 14 (Appendix B) lists some of the important pesticides and their cadmium content. Both fertilizers and pesticides are important to air pollution since they are intentionally introduced into the natural environment, usually in concentrations that can be hazardous to human, animal, and plant life.

A comparatively new and increasing use of cadmium is in cadmium-nickel batteries for civil and military aircraft, guided missiles, and spacecraft, as well as for portable electrical appliances and power tools.

Cadmium metal and cadmium nitrate are used to control the rate of nuclear fission and as reactor poison; cadmium soap (stearate) is used as a stabilizer of polyvinyl chloride; and diethyl cadmium is used in the production of tetraethyl lead.[97] One of the newest uses of cadmium is in semiconductors.

A listing of companies engaged in the production of cadmium chemicals appears in Table 15 in Appendix B.

No data indicating the emission rate of cadmium into the atmosphere from industrial users of cadmium products were found. The following is a list of activities which are probable sources of emission:

1. Manufacture of cadmium-faced bearings[45]
2. Production of alkaline batteries[32-34-67,72]
3. Casting of copper-cadmium alloys[11]
4. Electroplating[34]

5. Use of cadmium pigments (paints)[79,81]
6. Smelting of scrap metal (containing cadmium)
7. Welding of cadmium-plated metal parts

The United States Public Health Service[39] reported, in 1964, the following occupations to be potentially hazardous as a result of cadmium exposure:

Alloy making
Aluminum-solder making
Cadmium compound collecting-bag cleaning
Cadmium compound collecting-bag handling
Cadmium plating
Cadmium smelting
Cadmium vapor-lamp making
Cadmium working
Ceramic making
Dental-amalgam making
Electric-instrument making
Electroplating
Engraving
Glassmaking
Incandescent-lamp making
Lithography
Lithopone working
Metallizing
Paint making
Paint spraying
Photoelectric-cell making
Pigment making
Small-arms ammunition making
Smoke-bomb making
Soldering
Solder making
Storage-battery making
Textile printing
Welding, cadmium alloy
Welding, cadmium-plated objects
Zinc refining

3.4 Environmental Air Concentrations

Measurements of cadmium concentration in the air have been made by the National Air Sampling Network of the U.S. Public Health Service.[2,3] Air Quality Data figures reveal

that the average atmospheric concentration of cadmium for 35 stations in the United States was 0.002 $\mu g/m^3$ in 1964, and the maximum value recorded for this period was 0.350 $\mu g/m^3$. Tabulated data for these stations are contained in Table 16 in Appendix B.

4. ABATEMENT

No information was found in the literature on methods of abatement peculiar to air pollution by cadmium or its compounds. General control procedures for the prevention of air pollution by dusts, fumes, and mists applicable to the metal refinery, alloying, and machining industries are considered suitable to these processes in the cadmium industry.

Robertson[83] reported that in 1957 stack dust burden was reduced in a Canadian copper mining and smelting company when bag filters and cyclones were added to increase the recovery of cadmium as a by-product. A flue revision which vented the dryer gas through cyclones, separated the roaster and reverberating gas streams, and routed each stream through two existing electrostatic precipitators was supplementary to a baghouse installation (12 parallel units each consisting of a cooling train, a dust arrestor, and a fan). The flue system routed the furnace gases from the electrostatic precipitators to the filtration equipment (glass cloth) and discharged the filtered gas to the smelter stack. As a result of the added processes, more than 35 tons/day of dust containing 1.46 percent cadmium were collected.

5. ECONOMICS

No information has been found on the economic costs of cadmium air pollution or on the costs of its abatement.

Data on the production and consumption of cadmium are presented in Section 3.

6. METHODS OF ANALYSIS

6.1 Sampling Methods

Dusts and fumes of cadmium compounds may be collected by any method suitable for collection of other dusts and fumes; the impinger, electrostatic precipitator, and filter are commonly used. The National Air Sampling Network uses a high volume filtration sampler.[98]

6.2 Quantitative Methods

Emission spectroscopy has been used by the National Air Pollution Control Administration for cadmium analysis of samples from the National Air Sampling Network.[1,98] The samples are ashed and extracted to eliminate interfering elements. The minimum detectable cadmium concentration by emission spectroscopy is 0.011 $\mu g/m^3$ for urban samples and 0.004 $\mu g/m^3$ for nonurban samples. The different sensitivities result from the different extraction procedures required for urban samples.[4]

Thompson *et al.*[98] have reported that the National Air Pollution Control Administration uses atomic absorption to supplement analyses obtained by emission spectroscopy. The method has a minimum detectable limit of 0.0002 $\mu g/m^3$ based on a 2,000 m^3 air sample.

Emission spectroscopy has also been used to determine cadmium and other trace materials in mammalian tissues.[14] A resolution of 9,350 µg/100 g dry tissue was reported.

Polarographic methods[28,59] have also been used to determine cadmium concentrations in air. Ashing of the sample is followed by neutralization and precipitation of cadmium sulfate. The sample is recorded using a polarographic radiometer. This procedure has also been used to determine the presence of cadmium in urine, providing a measure of concentrations between 10,000 to 70,000 μg/liter.

A colorimetric method[86,93] was used to determine the concentration of cadmium in the air in a cadmium sterate plant. Dithizone was the coloring agent. The method has a sensitivity of 50 μg for cadmium if less than 10,000 μg of interfering metals are present.

7. SUMMARY AND CONCLUSIONS

Cadmium and cadmium compounds are toxic substances by all means of administration, producing acute or chronic symptoms varying in intensity from irritations to extensive disturbances resulting in death. However, despite increasing use of this metal and increasing attention to its toxic nature, the exact manner in which it affects human or animal organisms is not yet known. Cadmium is toxic to practically all systems and functions of the body, and is absorbed without regard to the levels of cadmium already present, thereby indicating the lack of a natural homeostatic mechanism for the control of organic concentrations of cadmium.

Inhalation of cadmium fumes, oxides, and salts often produces emphysema, which may be followed by bronchitis. Prolonged exposures to airborne cadmium frequently cause kidney damage resulting in proteinuria. Cadmium also affects the heart and liver. Statistical studies of people living in 28 U.S. cities have shown a positive correlation between heart diseases and the concentration of cadmium in the urban air. Cadmium may also be a carcinogen. While there is little evidence to support this conclusion from studies of industrial workers, animal experiments have shown cadmium may be carcinogenic. No data were found that indicated deleterious effects produced by airborne cadmium on commercial or domestic animals. However, experiments with laboratory animals have shown that cadmium affects the kidneys, lungs, heart, liver, and gastro-

intestinal organs, and the nervous and reproductive systems. No data were found on the effects of cadmium air pollution on plants or materials.

The metals industry is the major source of emissions of cadmium into the atmosphere. Cadmium dusts and fumes are produced in the extraction, refining, and processing of metallic cadmium. Since cadmium is generally produced as a by-product in the refining of other metals, such as zinc, lead, and copper, plants refining these materials are sources of cadmium emissions as well as of the basic metal. Also, because cadmium is present in small quantities in the ores of these metals, cadmium emissions may occur inadvertently in the refining of the basic metal.

Common sources of cadmium air pollution occur during the use of cadmium. Electroplating, alloying, and use of cadmium in pigments can produce local contaminations of the atmosphere. Also, since cadmium is added to pesticides and fertilizers, the use of these materials can cause local air pollution.

In 1964, the average concentration of cadmium in the ambient air was 0.002 $\mu g/m^3$, and the maximum concentration was 0.350 $\mu g/m^3$.

Air pollution control procedures are employed at some metal refinery plants in order to recover the valuable cadmium that would otherwise escape into the atmosphere. Electrostatic precipitators, baghouses, and cyclones are effectively used

for abatement. However, little information has been found on the specific application of these procedures for the purpose of controlling cadmium air pollution. The procedures for recovering cadmium from exhaust in a copper extraction plant collected significant quantities of valuable cadmium, at the same time reducing local air pollution levels.

No information has been found on the economic costs of cadmium air pollution or on the costs of its abatement. Methods are available for the analysis of cadmium in the ambient air.

Based on the material presented in this report, further studies are suggested in the following areas:

(1) Research into the effects of cadmium on human, animal, and plant health.

(2) Research into the carcinogenic effects of cadmium.

(3) Quantitative analysis of the emissions of cadmium into the atmosphere from industrial sources.

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APPENDIX A

APPENDIX A

PROPERTIES OF CADMIUM AND ITS COMPOUNDS[27,36]

A better understanding of cadmium's health effects requires knowledge of its properties. The physicochemical properties of cadmium and its compounds that may relate to their effects as air pollutants are given below.

1. Cadmium Metal

A. Physical Properties

Density (in g/cm^3)	
at p=1 atm	8.63-8.69
at absolute zero	8.65
below melting point	8.38
at 320°C	8.02
at 800°C	7.50
Vapor Density (in g/liter)	
at 320°C	0.0003
400°C	0.0033
500°C	0.032
600°C	0.175
700°C	0.66
766°C	1.36

Relative to air: 4.0

Melting Point	320.92°C
Boiling Point	766.0 °C

Vapor Pressure: variation with respect to temperature is given by the following two formulas:

$$\log P\ (mm) = -\frac{6,073}{T} - 0.9 \log T + 11.72 \quad (127\text{-}321^{\circ})$$

$$\log P\ (mm) = -\frac{5,982}{T} - 1.8 \log T + 14.063 \quad (321\text{-}877^{\circ})$$

Vapor Pressure at 200°C:	0.0003	mm mercury
at 800°C:	1,080.0	mm mercury

Cadmium is nonatomic in the vapor phase.

(continued)

APPENDIX A (Continued)

B. Chemical Properties

Group in Mendeleev's periodic table	II
Atomic weight	112.41
Atomic number	48
Valency: divalent	

Oxidation: in moist air, cadmium vapor reacts with water vapor to form cadmium oxide. Cadmium powder burns in the air with a red flame.

Solubility: cadmium dissolves in mercury, forming an amalgam. The solubility in mercury is 3.3 percent at 0°C; 5.9 percent at 25°C; and 24 percent at 100°C. Cadmium comes between iron and thorium in the electromotive series, thus aluminum, zinc, and iron displace cadmium from solutions of its salts; but cadmium displaces copper and other nobler metals from their solutions.

2. Cadmium Oxide (CdO)

Cadmium oxide can be prepared by the oxidation of cadmium metal by the air in the presence of cadmium vapor or the oxidation of its sulfide.

Melting point:* at atmospheric pressure it sublimes without melting at 1,770°K.

Vapor pressure: variation with temperature at

950°C=-3.15	3.385 (log P atm.)
1250°C=-1.43	1.404 (log P atm.)
(experimental)	(calculated)

Noticeable evaporation of cadmium oxide is observed at 900°C to 1,000°C.

Solubility in water: nearly insoluble

(continued)

APPENDIX A (Continued)

3. Cadmium Sulfide (CdS)

Vapor pressure variation with temperature is given by the formula:

$$\log P \text{ (mm)} = 9.823 - \frac{11,256}{T}$$

Some actual values are given below:

Temperature, °C	540	605	680	765	875	1,000	1,160
Pressure, mm Hg	10^{-4}	10^{-3}	10^{-2}	10^{-1}	1	10	100

Solubility in water: 1.5×10^{-10} mole/liter

Crystal dimensions of minerals:

Greenockite
Hexagonal a = 4.10 Å c = 6.69 Å
Sphalerite
Cubic a = 5.82 Å

On heating to 700°-800°C, the hexagonal forms reduce to the cubic form, and cadmium sulfide becomes brown and then carmine red.

4. Cadmium Sulfate ($CdSO_4$)

Density 4.96
Melting Point 1,000°C
Solubility in water:

0°C-74.5°C 76.48-87.62g $CdSO_4$/100 g water ($3CdSO_4 \cdot 8H_2O$)

77.0°C-100°C 73.02-60.77g $CdSO_4$/100 g water ($CdSO_4 \cdot H_2O$)

5. Cadmium Chloride ($CdCl_2$)

Density anhydrous 4.05
at mp 3.345

Crystal (hexagonal) lattice dimensions

a = 3.85 Å c = 17.46 Å

Melting Point* 564°-568°C
Melting Point* 964°-970°C

(continued)

APPENDIX A (Continued)

Vapor Pressure: variation with temperature

at 618°C	3 mm Hg
721	23
864	316
930	610

Solubility in water: numerous hydrates can be obtained.

A saturated aqueous solution of cadmium chloride contains the following amounts of cadmium chloride per 100 grams of water:

$CdCl_2$,	°C	0	30	100
	g	90	128.6	147

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*Data for melting and boiling points are slightly different from those stated in Table 12.

APPENDIX B

APPENDIX B

TABLE 10

EFFECTS OF CADMIUM AND CADMIUM COMPOUNDS ON HUMANS

Substance	Concentration	Exposure	Effects	Reference
Cadmium metal	530 ppm	Single dose	Cadmium gelatin produced sickness in children	40
Cadmium metal	67 ppm	Single dose	Cadmium in punch produced sickness in children	40
Cadmium metal	13-15 ppm		Cadmium in popsicles produced sickness in children	40
Cadmium oxide	11,000 $\mu g/m^3$	120 minutes	Retention of cadmium by kidneys, liver, pancreas, brain, bone marrow, lung, testes, myocardium, thyroid, adrenal cortex, and skin; very little elimination in 16 days	58
Cadmium oxide	8,630 $\mu g/m^3$	2,589 minutes	Lethal	10
Cadmium oxide	2,500,000-2,900,000 $\mu g/m^3$	1 minute	Lethal	29
Cadmium oxide	600-2,800 $\mu g/m^3$	13-38 years	Lethal	77
Cadmium oxide	>326,000 µg		Lethal	80
Cadmium sulfide	500,000 µg/kg wet wt		Lethal	80
Cadmium sulfide	165,000-97,600 $\mu g/m^3$	2-3 days	Anemia, giddiness, breathlessness, nausea, and fatigue	85

APPENDIX B

TABLE 11

PRESENCE OF CADMIUM IN HUMAN TISSUES[91]

Tissue	Cases			Amount of Cadmium Concentration (μg/g)
	Number Investigated	Number With Cadmium	Percent With Cadmium	
Muscle				
Diaphragm	91	7	8	
Other striated muscle	137	2	1	
Uterus	32	16	50	
Bladder	112	30	27	<50
Gastrointestinal tract				
Esophagus	67	6	9	
Stomach	130	41	31	
Duodenum	68	56	82	50
Jejunum	103	58	56	50
Ileum	84	25	30	
Cecum	31	16	51	
Omentum	73	42	58	
Sigmoid flexure of the colon	108	23	21	
Rectum	42	12	30	
Upper respiratory tract				
Larynx	48	2	4	<50
Trachea	60	12	20	
Genital Tract				
Ovary	16	31		
Testes	71	15	21	
Prostate	50	40	80	
Other				
Skin	21	0	0	
Thyroid gland	21	67		50
Adrenal gland	15	73		50
Fat	28	100		39
Bone	91	1	1	< 5

APPENDIX B

TABLE 12

PROPERTIES, TOXICITY, AND USES OF SOME CADMIUM COMPOUNDS[64]

Compound	Properties	Toxicity	Uses
Cadmium acetate $Cd(CH_3COO)_2$	mp anhydrous 255°C	Ingestion (metal and soluble compounds): increased salivation, choking, vomiting, abdominal pain, diarrhea, tenesmus. Inhalation (dust and fumes): throat dryness, cough, headache, vomiting, chest pain, irritability, pneumonitis	For producing iridescent effect on porcelains and pottery; as a reagent for determination of S, Se, and Te in cadmium electroplating
Cadmium bromide $CdBr_2$	mp 566° bp 963°	Same as above	In photography, process engraving, and lithography
Cadmium carbonate $CdCO_3$	Decomposes < 500	Same as above	
Cadmium chloride $CdCl_2$	mp 568° bp 960°	Same as above	In photography; in dyeing and calico printing; in the vacuum tube industry; in manufacture of cadmium yellow; special mirrors; as ice-nucleating agent, lubricant; in analysis of sulfides to absorb the H_2S

(continued)

APPENDIX B

TABLE 12 (Continued)

PROPERTIES, TOXICITY, AND USES OF SOME CADMIUM COMPOUNDS

Compound	Properties	Toxicity	Uses
Cadmium cyanide $Cd(CN)_2$	Decomposes >200°	Very poisonous	In copper bright electroplating
Cadmium fluoride CdF_2	mp 1049° bp 1748°	Poisonous	In manufacture of phosphors, glass; in nuclear reactor controls
Cadmium hydroxide $Cd(OH)_2$	Decomposes 300°	Same as Cadmium acetate	In storage battery electrodes
Cadmium iodide CdI_2	mp 388° bp 787°	Same as Cadmium acetate	In electrodeposition of Cd; as nematocide; in manufacture of phosphors; as lubricant; in photoconductors; in photography, process engraving, lithography
Cadmium nitrate $Cd(NO_3)_2$	mp 59.5° bp 132°		In making other Cd salts; in photographic emulsions

(continued)

APPENDIX B

TABLE 12 (Continued)

PROPERTIES, TOXICITY, AND USES OF SOME CADMIUM COMPOUNDS

Compound	Properties	Toxicity	Uses
Cadmium oxide CdO	mp >1426° bp, Decomposes 900-1000°	Poisonous. Acute poisoning occurs in animals at the following concentrations of cadmium oxide (in $\mu g/m^3$ of air inhaled): for rats - 500,000 for rabbits - 2,500,000 for dogs - 4,000,000 for monkeys - 1,500,000. For humans, inhalation of air containing 2,500,000 $\mu g/m^3$ for one minute or air containing 5,000,000 $\mu g/m^3$ for ½ minute is fatal. Also see Cadmium acetate	In phosphorus, semiconductors, manufacture of silver alloys, glass; in storage battery electrodes; as nematocide; as catalyst for organic reactions; in cadmium electroplating; in ceramic glazes. Vet. use: as an ascaricide in swine
Cadmium potassium cyanide $K_2Cd(CN)_4$	mp 450°	Poisoning may occur by ingestion, absorption through injured skin	
Cadmium salicylate $Cd(C_7H_5O_3)_2$	mp 242°		Med. use: has been used as an antiseptic

(continued)

APPENDIX B

TABLE 12 (Continued)

PROPERTIES, TOXICITY, AND USES OF SOME CADMIUM COMPOUNDS

Compound	Properties	Toxicity	Uses
Cadmium selenide CdSe	mp 1350°	Low toxicity because of insolubility	In photoconductors, semiconductors, photoelectric cells, and rectifiers; in phosphors
Cadmium succinate $C_4H_4CdO_4$			As plant fungicide
Cadmium sulfate $CdSO_4$	mp 1000°	LD in dogs 27,000 µg/kg	In electrodeposition of Cd, Cu, and Ni; in phosphors; in manufacture of standard cadmium elements
Cadmium sulfide CdS	mp 1750[100] atm bp sublimes in N_2		As a pigment that is fast to light, and not affected by H_2S; as color for soaps: for coloring glass yellow, coloring textiles, paper, rubber; in printing inks, ceramic glazes, fireworks. Med. use: for seborrheic dermatitis
Cadmium telluride CdTe	mp 1041°		In semiconductor research, in phosphors
Cadmium tungstate $CdWO_4$			In X-ray screens; in scintillation counters; in phosphors; as catalyst for organic reactions

(continued)

APPENDIX B

TABLE 12 (Continued)

PROPERTIES, TOXICITY, AND USES OF SOME CADMIUM COMPOUNDS

Compound	Properties	Toxicity	Uses
Dimethylcadmium $Cd(CH_3)_2$	mp 4.5° Decomposes with explosive violence when heated above 150°. Catches fire when dropped on filter paper and produces dense clouds of first white, then brown cadmium oxide smoke		In organic synthesis; as polymerization catalyst
Potassium tetraiodocadmate $K_2[CdI_4]\ 2H_2O$		See Cadmium acetate	In testing for alkaloids, amines, imines, heterocyclic nitrogens, peroxides in ether

Note: More detailed information was not available on the following compounds:

Cadmium borate	$Cd(BO_2)_2 \cdot xH_2O$	Cadmium perchlorate	$Cd(ClO_4)_2 \cdot 6H_2O$
Cadmium bromate	$Cd(BrO_3)_2 \cdot H_2O$	Cadmium phosphate	$Cd_3(PO_4)_2$
Cadmium bromide anhydrous	$CdBr_2$	Cadmium phosphide	CdP
Cadmium chromate	$CdCrO_4$	Cadmium zirconate	$CdZrO_3$
Cadmium hexafluoroarsenate	$Cd(AsF_6)_2$	Potassium cadmium iodide	$K_2CdI_4 \cdot 2H_2O$
Cadmium molybdate	$CdMoO_4$	Potassium tetracyanocadmiate	$K_2Cd(CN)_4$

APPENDIX B

TABLE 13

EFFECTS OF CADMIUM AND CADMIUM COMPOUNDS ON ANIMALS

Substance	Subject	Concentration	Exposure	Effects	Reference
Cadmium metal	Rats	135 ppm	Single dose	Lethal	3
Cadmium metal	Rats	5-75 ppm	Single dose	Lethal	3
Cadmium metal	Rats	0.1-10 ppm	1 year	Cadmium content of liver and kidney increased in proportion to quantity of cadmium intake	3
Cadmium metal	Cats	20 ppm	Single dose	Produced vomiting	6
Cadmium oxide	Rats	500,000 $\mu g\text{-}min/m^3$	1 minute	Acute pulmonary edema within 24 hours; pneumonitis in 3-10 days; permanent lung damage and fibrosis	70
Cadmium oxide	Rats	150,000-1,300,000 $\mu g/min/m^3$	10-15 minutes	Acute pulmonary edema within 24 hours; pneumonitis in 3-10 days; permanent lung damage and fibrosis	8
Cadmium oxide	Rats	800,000-1,000,000 $\mu g\text{-}min/m^3$	1 minute	Lethal; acute pulmonary edema within 24 hours; pneumonitis in 3-10 days; permanent lung damage and fibrosis	70

(continued)

APPENDIX B

TABLE 13 (Continued)

EFFECTS OF CADMIUM AND CADMIUM COMPOUNDS ON ANIMALS

Substance	Subject	Concentration	Exposure	Effects	Reference
Cadmium oxide	Mice	660,000-1,130,000 $\mu g\text{-}min/m^3$	15 minutes		8
Cadmium oxide	Rabbits	630-3,690 $\mu g/m^3$	13-30 minutes		8
Cadmium oxide	Guinea pigs	640,000-6,450,000 $\mu g/m^3$	13-30 minutes		8
Cadmium oxide	Dogs	3,100,000-10,000,000 $\mu g/m^3$	10-20 minutes	Lethal	80
Cadmium oxide	Dogs	7,000$\mu g/m^3$	1,102 hours	Retention of cadmium dust in tissues	8
Cadmium oxide	Monkeys	4,500-28,200 $\mu g/m^3$	10-30 minutes	Lethal	8
Cadmium chloride	Mice	4,480 $\mu g/kg$ body wt.	1-7 days	Lethal	104
Cadmium chloride	Mice	85-170 $\mu g/liter$		Overall retention 10.5-23%	76
Cadmium chloride	Rats	78,000-164,000 $\mu g\text{-}min/m^3$	6 months	Tolerated	70
Cadmium chloride	Rats	250,000 $\mu g\text{-}min/m^3$	15 minutes	Lethal; pulmonary edema within 24 hours; pneumonitis 3-10 days; permanent lung damage and fibrosis	70

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APPENDIX B

TABLE 13 (Continued)

EFFECTS OF CADMIUM AND CADMIUM COMPOUNDS ON ANIMALS

Substance	Subject	Concentration	Exposure	Effects	Reference
Cadmium chloride	Rats	170-340 µg ions	Single dose	Pleomorphic sarcomata 10-16 months after injection; carcinogenicity observed only in tissue of mesenchymal mesodermal origin	41
Cadmium chloride	Rabbits	250 µg/kg body wt	12-29 weeks	Pulmonary edema	7
Cadmium chloride	Dogs	320 µg/liter	30 minutes	Lethal	46
Cadmium chloride	Dogs	9,600,000 µg-min/m^3	1 minute	Lethal	40
Cadmium stearate	Mice	9,310-18,570 µg/m^3/day	1-7 days	Lethal; LD 9,310 µg	104
Cadmium stearate	Mice	6,000-7,570 µg/m^3/day	1-7 days	Growth suppression, histological changes in stomach, intestine, testicles, other organs	104
Cadmium stearate	Rats	0.1-1% of food	90 days	Anemia, giddiness, breathlessness, nausea, and fatigue	85

APPENDIX B

TABLE 14

CADMIUM-CONTAINING PESTICIDES[31]

Use*	Trade Name	Chemical Ingredients		Supplier	
		Name	%	Name	Location
F/D	Stauffer Mer-Caid	Anilinocadmium dilactate	6.25	Stauffer Chem. Co.	New York
F/D	Twin Light Granular Lawn Fungicide	Cadmium chloride, Thiram		Seacoast Labs, Inc.	E. Brunswick, N.J.
F/S	Ortho Lawn Disease Control	Cadmium carbonate, captan, 5-chloron nitrobenzene	3.00	Chevron Chem. Co., Ortho Div.	Richmond, Calif.
F/S	Ortho Lawn and Turf Fungicide	Cadmium carbonate, Folpet, Thiram	5.00	Chevron Chem. Co., Ortho Div.	Richmond, Calif.
F/S	Caddy	Cadmium chloride	20.10	Cleary, W.A., Corp.	N. Brunswick, N.J.
F/S	Vicad	Cadmium chloride		Vineland Chem. Co.	Vineland, N.J.
F/S	Cad-Trete	Cadmium chlorate, hydrate, Thiram	8.30	Cleary, W.A., Corp.	N. Brunswick, N.J.
F/S	Cadminate	Cadmium, Cadmium succinate	29.00 60.00	Mallinckrodt Chemical Works	St. Louis, Mo.
F/S	Agway Fire Blight Canker Paint	Cadmium sulfate	14.00	Agway	

(continued)

APPENDIX B

TABLE 14 (Continued)

CADMIUM-CONTAINING PESTICIDES[31]

Use*	Trade Name	Chemical Ingredients		Supplier	
		Name	%	Name	Location
F/S	Ultra-Clor (General Turf Fungicide)	Cadmium, Cadmium succinate, Mercuric dimethyl-thiocarbamate, potas-sium chromate, mercury and chromium	1.10 3.30	Chevron Chem. Co.	Richmond, Calif.
F/S	Fura Turf	Phenylamine cadmium dilactate	12.80	Guard Chem. Co., Inc.	Ossining, N.Y.
F-I /S	Ortho Lawn Fungicide	Cadmium carbonate, Captan, Gamma (BHC)	5.30	Chevron Chem. Co.	Richmond Calif.

*F = Fungicides D = Dusts; S = Sprays; and I = Insecticides.

APPENDIX B

TABLE 15

LIST OF COMPANIES PRODUCING CADMIUM CHEMICALS (1968)[18]

1. CADMIUM ACETATE	
J. T. Baker Chem. Co.	Phillipsburg, N.J.
The Harshaw Chem. Co.	Cleveland, Ohio
Mallinckrodt Chem. Works Industrial Chem. Div.	St. Louis, Mo.
The Shepherd Chem. Co.	Cincinnati, Ohio
2. CADMIUM BENZOATE	
Carlisle Chem. Works, Inc.	Reading, Ohio
3. CADMIUM BISULFATE AND 4. CADMIUM BROMATE	
City Chem. Corp.	Jersey City, N.J.
5. CADMIUM BROMIDE	
The Dow Chemical Co.	Midland, Mich.
Research Inorganic Chem. Co.	Sun Valley, Calif.
6. CADMIUM CARBONATE	
Allied Chem. Corp. Industrial Chem. Div.	Marcus Hook, Pa.
Chemetron Corp. McGean Chem. Co., Div.	Cleveland, Ohio
The Shepherd Chem. Co.	Cincinnati, Ohio
7. CADMIUM CHLORIDE	
Allied Chem. Corp. Industrial Chem. Div.	Marcus Hook, Pa.
J. T. Baker Chem. Co.	Phillipsburg, N.J.
Chemetron Corp. McGean Chem. Co., Div.	Cleveland, Ohio
W. A. Cleary Corp.	New Brunswick, N.J.
The Harshaw Chem. Co.	Cleveland, Ohio
Mallinckrodt Chem. Works Industrial Chem. Div.	St. Louis, Mo.

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TABLE 15 (Continued)

8.	CADMIUM CHROMATE	
	City Chem. Corp.	Jersey City, N.J.
9.	CADMIUM CYANIDE	
	Aceto Chem. Co., Inc.	Flushing, N.Y.
10.	CADMIUM DICHROMATE	
	City Chem. Corp.	Jersey City, N.J.
11.	CADMIUM ETHYLENEDIAMINE	
	Allied Chem. Corp. Industrial Chem. Div.	Marcus Hook, Pa.
12.	CADMIUM 2-ETHYLHEXANOATE (CADMIUM OCTOATE)	
	Carlisle Chem. Works, Inc. Advance Div.	New Brunswick, N.J.
	The Shepherd Chem. Co.	Cincinnati, Ohio
	Tenneco Chem. Inc. Nuodex Div.	Elizabeth, N.J. Long Beach, Calif.
13.	CADMIUM FERROCYANIDE	
	City Chem. Corp.	Jersey City, N.J.
14.	CADMIUM FLUOBORATE	
	Allied Chem. Corp. Industrial Chem. Div.	Marcus Hook, Pa.
	Harstan Chem. Corp.	Brooklyn, N.Y.
	The Harshaw Chem. Co.	Cleveland, Ohio
15.	CADMIUM FLUORIDE	
	Allied Chem. Corp. Industrial Chem. Div.	Marcus Hook, Pa.
	The Harshaw Chem. Co. Crystal-Solid State Div.	Cleveland, Ohio Elyria, Ohio
	Semi-Elements, Inc.	Saxonburg, Pa.
	Sylvania Electric Products, Inc.	Towanda, Pa.
16.	CADMIUM FORMATE	
	City Chem. Corp.	Jersey City, N.J.

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APPENDIX B

TABLE 15 (Continued)

17. CADMIUM HYDROXIDE (CADMIUM HYDRATE)	
Chemetron Corp. McGean Chem. Co., Div.	Cleveland, Ohio
The Shepherd Chem. Co.	Cincinnati, Ohio
18. CADMIUM IODIDE	
Allied Chem. Corp. Industrial Chem. Div.	Marcus Hook, Pa.
J. T. Baker Chem. Co.	Phillipsburg, Pa.
Mallinckrodt Chem. Works Industrial Chem. Div.	St. Louis, Mo.
R.S.A. Corp.	Ardsley, N.Y.
19. CADMIUM LACTATE	
City Chem. Corp.	Jersey City, N.J.
20. CADMIUM LAURATE	
Tenneco Chem. Inc. Nuodex Div.	Elizabeth, N.J. Long Beach, Calif.
21. CADMIUM MOLYBDATE	
City Chem. Corp.	Jersey City, N.J.
Sylvania Electric Products, Inc.	Towanda, Pa.
22. CADMIUM NAPHTHENATE	
Carlisle Chem. Works, Inc. Advance Div.	New Brunswick, N.J.
The Harshaw Chem. Co.	Gloucester City, N.J.
The Shepherd Chem. Co.	Cincinnati, Ohio
Frederick A. Stresen-Reuter, Inc.	Bensenville, Ill.
Tenneco Chem. Inc. Nuodex Div.	Elizabeth, N.J. Long Beach, Calif.
23. CADMIUM NEO-DECANOATE	
The Shepherd Chem. Co.	Cincinnati, Ohio

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TABLE 15 (Continued)

24. CADMIUM NITRATE	
Allied Chem. Corp. Industrial Chem. Div.	Buffalo, N.Y.
J. T. Baker Chem. Co.	Phillipsburg, N.J.
Chemetron Corp. McGean Chem. Co., Div.	Cleveland, Ohio
The Harshaw Chem. Co.	Gloucester City, N.J.
Mallinckrodt Chem. Works Industrial Chem. Div.	St. Louis, Mo.
The Shepherd Chem. Co.	Cincinnati, Ohio
25. CADMIUM OXALATE	
Chemetron Corp. McGean Chem. Co., Div.	Cleveland, Ohio
City Chem. Corp.	Jersey City, N.J.
26. CADMIUM OXIDE	
Allied Chemical Corp. Industrial Chem. Div.	Marcus Hook, Pa.
American Smelting & Refining Co.	Denver, Colo.
J. T. Baker Chem. Co.	Phillipsburg, N.J.
Chemetron Corp. McGean Chem. Co., Div.	Cleveland, Ohio
The Harshaw Chem. Co.	Elyria, Ohio
27. CADMIUM POTASSIUM CHLORIDE	
City Chem. Corp.	Jersey City, N.J.
28. CADMIUM POTASSIUM CYANIDE	
City Chem. Corp.	Jersey City, N.J.
29. CADMIUM POTASSIUM IODIDE	
City Chem. Corp.	Jersey City, N.J.
30. CADMIUM PROPIONATE	
The Shepherd Chem. Co.	Cincinnati, Ohio
31. CADMIUM SALICYLATE	
City Chem. Corp.	Jersey City, N.J.

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APPENDIX B

TABLE 15 (Continued)

32. CADMIUM SEBACATE	
Mallinckrodt Chem. Works Industrial Chem. Div.	St. Louis, Mo.
33. CADMIUM SELENATE	
City Chem. Corp.	Jersey City, N.J.
34. CADMIUM SELENIDE (CADMIUM ORANGE)	
Allied Chem. Corp. Industrial Chem. Div.	Marcus Hook, Pa.
The Glidden Co.	Baltimore, Md.
Hercules Powder Co. Imperial Color & Chem. Dept.	Glen Falls, N.Y.
The Harshaw Chem. Co. Crystal-Solid State Div.	Cleveland, Ohio Elyria, Ohio
H. Kohnstamm and Co., Inc. General Color Co., Div.	Newark, N.J.
Research Inorganic Chem. Co.	Sunn Valley, Calif.
Semi-Elements, Inc.	Saxonburg, Pa.
35. CADMIUM m-SILICATE	
City Chem. Corp.	Jersey City, N.J.
36. CADMIUM SILICOFLUORIDE	
City Chem. Corp.	Jersey City, N.J.
37. CADMIUM STEARATE	
Argus Chem. Corp.	Brooklyn, N.Y.
Nopco Chem. Co. Industrial Div.	Cedartwon, Ga. Richmond, Calif.
S. B. Penick & Co. M. W. Parsons-Plymouth Div.	Newark, N.J.
Synthetic Products Co.	Cleveland, Ohio
Witco Chem. Co., Inc. Organic Chem. Div.	Chicago, Ill. Los Angeles, Calif. Perth Amboy, N.J.

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TABLE 15 (Continued)

38. CADMIUM SULFATE	
Allied Chem. Corp.	
Industrial Chem. Div.	Marcus Hook, Pa.
J. T. Baker Chem. Co.	Phillipsburg, N.J.
Chemetron Corp.	
McGean Chem. Co., Div.	Cleveland, Ohio
The Harshaw Chem. Co.	Cleveland, Ohio
Mallinckrodt Chem. Works	
Industrial Chem. Div.	St. Louis, Mo.
The Shepherd Chem. Co.	Cincinnati, Ohio
39. CADMIUM SULFIDE (CADMIUM YELLOW)	
Allied Chem. Corp.	
Industraal Chem. Div.	Marcus Hook, Pa.
American Smelting and Refining Co.	Denver, Colo.
The Eagle-Picher Co.	Miami, Okla.
The Glidden Co.	Baltimore, Md.
Hercules Powder Co.	
Imperial Color & Chem. Dept.	Glen Falls, N.Y.
The Harshaw Chem. Co.	
Crystal-Solid State Div.	Cleveland, Ohio
	Elyria, Ohio
H. Kohnstamm and Co., Inc.	
General Color Co., Div.	Newark, N.J.
Research Inorganic Chem. Co.	Sun Valley, Calif.
Semi-Elements, Inc.	Saxonburg, Pa.
Smith Chem. & Color Co., Inc.	Brooklyn, N.Y.
Sylvania Electric Products Inc.	
Chem. and Metallurgical Div.	Towanda, Pa.
40. CADMIUM SULFOSELENIDES (CADMIUM REDS)	
B. F. Drakenfeld and Co., Inc.	Washington, Pa.
The Glidden Co.	Baltimore, Md.
Hercules Powder Co.	
Imperial Color & Chem. Dept.	Glen Falls, N.Y.
H. Kohnstamm and Co., Inc.	
General Color Co., Div.	Newark, N.J.
Smith Chem. & Color Co., Inc.	Brooklyn, N.Y.
41. CADMIUM TARTRATE	
City Chem. Corp.	Jersey City, N.J.

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TABLE 15 (Continued)

42. CADMIUM TELLURIDE	
City Chem. Corp.	Jersey City, N.J.
The Eagle-Picher Co.	Miami, Okla.
The Harshaw Chem. Co. Crystal-Solid State Div.	Cleveland, Ohio
	Elyria, Ohio
Semi-Elements, Inc.	Saxonburg, Pa.
43. CADMIUM THIOCYANATE	
City Chem. Corp.	Jersey City, N.J.
44. CADMIUM TUNGSTATE	
North Metal and Chem. Co.	York, Pa.
Semi-Elements, Inc.	Saxonburg, Pa.
Sylvania Electric Products, Inc.	Towanda, Pa.
45. CADMIUM ZIRCONATE	
National Lead Co. Titanium Alloy Manufacturing Div.	Niagara Falls, N.Y.
Research Inorganic Chem. Co.	Sun Valley, Calif.
46. CADMIUM SUCCINATE (Pesticide)	
Mallinckrodt Chem. Works	St. Louis, Mo.

APPENDIX B

TABLE 16

CONCENTRATION OF CADMIUM IN THE AIR[2,3]
($\mu g/m^3$)

	1954-59		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Alabama												
Birmingham	.018										.029	
Huntsville										.000		
Arizona												
Phoenix			.068	.016			.000				.020	
Tucson			.140	.026								
California												
Los Angeles	.020		.029	.009					.022			
Pasadena								.003				
San Francisco							.051		.008			
Santa Barbara								.000				
Colorado												
Denver	.087						.310		.065		.030	
Pueblo										.006		
Connecticut												
Norwich										.000		
Waterbury			.045	.015								
District of Columbia												
Washington							.048		.028		.009	
Florida												
St. Petersburg								.000				
Georgia												
Atlanta	.044								.020			
Idaho												
Boise			.000				.000				.000	

(continued)

APPENDIX B

TABLE 16 (Continued)

CONCENTRATION OF CADMIUM IN THE AIR
($\mu g/m^3$)

	1954-59		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Illinois												
Chicago	.017		.400	.033	.150							
Cicero							.047		.054		.049	
East St. Louis												
Joliet									.180			
North Chicago										.000		
Springfield										.000		
Indiana								.000				
East Chicago												
Gary			.110	.018							.042	
Indianapolis	.040		.100	.029	.029							
Muncie												
Iowa										.000		
Cedar Rapids												
Des Moines	.009		.017							.000		
Louisiana							.000				.000	
Lake Charles												
New Orleans	.019							.000				
Maryland							.010		.000			
Baltimore	.034		.044									
Cumberland											.110	
Massachusetts										.000		
Boston												
Michigan											.000	
Detroit	.051											
Grand Rapids							.042		.017		.000	
Minnesota										.000		
Minneapolis	.027											
St. Paul								.000				

(continued)

APPENDIX B

TABLE 16 (Continued)

CONCENTRATION OF CADMIUM IN THE AIR
($\mu g/m^3$)

	1954-59		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Missouri												
St. Louis	.150						.420		.051		.290	
Montana												
Helena											.049	
Nebraska												
Omaha					.110	.011						
Nevada												
Las Vegas					.000		.000		.000		.000	
Reno										.000		
New Mexico												
Albuquerque			.018	.003								
New Jersey												
Bayonne										.028		
Camden								.011				
Newark					.310	.056					.350	
Paterson										.002		
New York												
Buffalo							.011					
Glen Cove								.005				
Massena						.000						
Mount Vernon						.000						
New Rochelle					.	.003						
New York					.210	.032	.180		.064		.040	
Rochester						.000						
Troy								.000				
North Carolina												
Asheville				.000								
Charlotte											.000	
Durham										.000		

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APPENDIX B

TABLE 16 (Continued)

CONCENTRATION OF CADMIUM IN THE AIR
($\mu g/m^3$)

	1954-59		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Ohio												
Akron			.130	.020								
Canton					.095	.023						
Cincinnati					.093	.021			.036		.110	
Cleveland							.037		.064		.038	
Columbus					.110	.026						
Dayton			.019									
Youngstown			.140	.024								
Oklahoma												
Tulsa						.000						
Oklahoma City					.045	.015						
Oregon												
Eugene					.000							
Medford					.013	.001						
Portland	.028											
Pennsylvania												
Allentown					.330	.068						
Altoona										.003		
Bethlehem					.140	.039						
Lancaster								.000				
Philadelphia	.016				.290	.045			.046		.110	
Pittsburgh			.067				.095		.065		.028	
Scranton					.089	.024						
Rhode Island												
East Providence										.000		
Tennesse												
Chattanooga					.039	.006						
Nashville	.016										.014	

(continued)

APPENDIX B

TABLE 16 (Continued)

CONCENTRATION OF CADMIUM IN THE AIR
($\mu g/m^3$)

	1954-59		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Texas												
El Paso								.310			.300	
Houston	.026											
Odessa										.000		
Tyler										.000		
Utah												
Salt Lake City	.028											
Washington												
Seattle	.011						.032		.000			
Spokane						.000						
Tacoma	.170						.000				.000	
West Virginia												
Charleston	.071	.015			.160	.043					.000	
Huntington				.021								
Wisconsin												
Milwaukee	.043										.000	
Wyoming												
Cheyenne											.000	